CLAIMS

1. A non-human animal having a neurologic disease induced by the process of:

perfusing the non-human animal with a pharmacologically effective amount of a combination of an Aβ compound, at least one pro-oxidative compound, and at least one anti-oxidant inhibitor, wherein the perfusion produces impaired performance of the animal in memory and learning tests and induces abnormal neuropathology in a brain of the animal, wherein said impaired performance and abnormal neuropathology are in comparison with control non-human animals.

2. The non-human animal of claim 1, wherein the Aβ compound comprises $A\beta_{42}$.

3. The non-human animal of claim 1, wherein the Aβ compound comprises a peptide fragment of $A\beta_{42}$.

4. The non-human animal of claim 3, wherein the peptide fragment of $A\beta_{42}$ comprises at least one of $A\beta_{1-40}$ or $A\beta_{24-35}$.

5. The non-human animal of claim 1, wherein the Aβ compound comprises a peptidomimetic that mimicks $A\beta_{42}$.

6. The non-human animal of claim 1, wherein the at least one pro-oxidative compound is selected from the group consisting of ferrous sulfate, copper sulfate, cobalt sulfate, manganese sulfate, and zinc sulfate.

7. The non-human animal of claim 1, wherein the at least one pro-oxidative compound comprises ferrous sulfate.

8. The non-human animal of claim 1, wherein the at least one anti-oxidant inhibitor comprises buthionine sulfoximine.

9. The non-human animal of claim 1, wherein the process further comprises perfusing the non-human animal with an effective amount of a phosphatase inhibitor.

10. The non-human animal of claim 9, wherein the phosphatase inhibitor is selected from the group consisting of okadaic acid, 1-nor-okadaone, bioallethrin, calycullin A, cantharidic acid, cantharidin, cypermethrin, deltamethrin, endothall, endothall thioanhydride, fenvalerate, okadol, permethrin, phenylarsine oxide, pyrophosphate, sodium fluoride, and vanadate.

11. The non-human animal of claim 9, wherein the phophatase inhibitor comprises okadaic acid.

12. The non-human animal of claim 1, wherein the process further comprises perfusing the non-human animal with an effective amount of a pro-inflammatory compound.

13. The non-human animal of claim 12, wherein the pro-inflammatory compound is selected from the group consisting of TNF-α, IL-6, and IL-1b.

14. The non-human animal of claim 12, wherein the pro-inflammatory compound comprises TNF-α.

15. A method for inducing a neurologic disease in a non-human animal, comprising:

perfusing the non-human animal with a pharmacologically effective amount of a combination of an Aβ compound, at least one pro-oxidative compound, and at least one anti-oxidant inhibitor.

16. The method of claim 15, wherein the Aβ compound comprises $A\beta_{42}$.

17. The method of claim 15, wherein the Aβ compound comprises a peptide fragment of $A\beta_{42}$.

18. The method of claim 17, wherein the peptide fragment of $A\beta_{42}$ comprises at least one of $A\beta_{1-40}$ or $A\beta_{24-35}$.

19. The method of claim 15, wherein the Aβ compound comprises a peptidomimetic that mimicks $A\beta_{42}$.

20. The method of claim 15, wherein the at least one pro-oxidative compound is selected from the group consisting of ferrous sulfate, copper sulfate, cobalt sulfate, manganese sulfate, and zinc sulfate.

21. The method of claim 15, wherein the at least one pro-oxidative compound comprises ferrous sulfate.

22. The method of claim 15, wherein the at least one anti-oxidant inhibitor comprises buthionine sulfoximine.

23. The method of Claim 15, further comprising perfusing the non-human animal with an effective amount of a phosphatase inhibitor.

24. The method of claim 23, wherein the phosphatase inhibitor is selected from the group consisting of okadaic acid, 1-nor-okadaone, bioallethrin, calycullin A, cantharidic acid, cantharidin, cypermethrin, deltamethrin, endothall, endothall thioanhydride, fenvalerte, okadol, permethrin, phenylarsine oxide, pyrophosphate, sodium fluoride, and vanadate.

25. The method of claim 23, wherein the phophatase inhibitor comprises okadaic acid.

26. The method of claim 15, further comprising perfusing the non-human animal with an effective amount of a pro-inflammatory compound.

27. The method of claim 27, wherein the pro-inflammatory compound is selected from the group consisting of TNF-α, IL-6, and IL-1b.

28. The method of claim 27, wherein the pro-inflammatory compound comprises TNF-α.

29. A method of screening for an agent that ameliorates symptoms of a neurologic disease, said method comprising:

comparing performance on memory and learning tests of a first non-human animal contacted with the agent with that of a second non-human animal not contacted with the agent, wherein the first and said second non-human animals have been co-

infused with a pharmacologically effective amount of Aβ, at least one pro-oxidative compound, and at least one anti-oxidant inhibitor wherein the co-infusion produces impaired performance on the memory and learning tests and abnormal neuropathology in a brain of the first and second non-human animals, wherein the impaired performance and the abnormal neuropathology are in comparison with control non-human animals, whereby an agent which ameliorates the symptoms is identified by superior performance of said first non-human animal in comparison with the second non-human animal on the memory and learning tests.

30. A method for screening for an agent useful for treating a neurologic disease, said method comprising:

comparing performance on memory and learning tests of a first non-human animal contacted with the agent with that of a second non-human animal not contacted with the agent, wherein the first and said second non-human animals have been co-infused with a pharmacologically effective amount of Aβ and at least one pro-oxidative compound, and at least one anti-oxidant inhibitor, wherein the co-infusion produces impaired performance on the memory and learning tests and abnormal neuropathology in a brain of the first and second non-human animals, wherein the impaired performance and the abnormal neuropathology are compared with control non-human animals; and comparing neuropathology in the brain of the first and the second non-human animal when said first non-human animal exhibits superior performance on the memory and learning tests compared with the second non-human animal, whereby an agent which is useful for treating a neurologic disease is identified by a decrease in neuropathologic findings in the first non-human animal in comparison with the second non-human animal.